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1: *Arterioscler Thromb Vasc Biol* 2000 Feb;20 (2):435-42

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Complete atherosclerosis regression after human ApoE gene transfer in ApoE-deficient/nude mice.

Desurmont C, Caillaud JM, Emmanuel F, Benoit P, Fruchart JC, Castro G, Branellec D, Heard JM, Duverger N

Laboratoire RTG, Institut Pasteur, Paris, France.

The apolipoprotein E (apoE)-deficient mouse is a relevant animal model of human atherosclerosis. Although the prevention of atherosclerosis development has been documented after somatic gene transfer into animal models, regression of lesions remains to be demonstrated. Thus, we used this genetically defined mouse model nn the nude background to show atherosclerosis regression. ApoE-deficient nude mice were infected with 5 x 10(8) or 10(9) plaque-forming units of a first-generation adenovirus encoding human apoE cDNA. The secretion of human apoE resulted in a rapid decrease of total cholesterol, which normalized the hypercholesterolemic phenotype within 14 days (from 600+/-100 to <100 microg/mL). Transgene expression was observed during a period of >4 months, with a normalization of cholesterol and triglyceride levels during 5 months. At that time, we successfully reinjected the recombinant adenovirus and observed the appearance of the human protein as well as the correction of lipoprotein phenotype. In mice killed 6 months-after the first infection, we observed a dose-dependent regression of fatty streak lesions in the aorta. We showed sustained expression of a transgene with a first-generation adenoviral vector and a correction of dyslipoproteinemia phenotype leading to lesion regression. These data demonstrate that somatic gene transfer can induce plaque regression.

PMID: 10669641, UI: 20136123

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1: *Curr Opin Lipidol* 2000 Feb;11(1):25-9 Related Articles, Books, LinkOut

The value of apolipoprotein E knockout mice for studying the effects of dietary fat and cholesterol on atherogenesis.

Osada J, Joven J, Maeda N

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The ability of the apolipoprotein E-deficient mouse to develop spontaneous atherosclerosis, which resembles the human process, is an excellent model in which to assess the impact of dietary factors. This review discusses the role of several nutrients in the development of atherosclerosis and the mechanisms through which they act.

Publication Types:
- Review
- Review, tutorial

PMID: 10750690, UI: 20212728